THE DISTINCTIVE ELECTROCARDIOGRAM OF CORONARY ARTERIOSPASM

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The value of the electrocardiogram in the diagnosis of cardiac pain needs no emphasis. Experience has confirmed the unreliability of the patient's description of his pain as a guide to its cause. Thus, when the progress of over 300 patients in whom chest pain was subjectively characteristic of cardiac pain, was watched over many years, the pain proved to have a dyspeptic or some other non-cardiac source, and successive electrocardiograms remained normal (Evans, 1952). The history of the illness in such patients differed in no particular way from that obtained from a patient where the cardiogram proved the presence of cardiac infarction. Thus, the pain, usually in the centre of the chest, was described as gripping or tightness, often radiating into the arms or the jaw, and readily brought on by exertion, and as a rule yielding as quickly to rest. It is also known that physical signs are sparse in the many patients in whom the myocardial injury initiating the pain is not severe enough to prevent them from being ambulatory or even from following their customary occupation. In more than four-fifths of such cases reliance has to be placed on the electrocardiogram as the only means of telling the cause of the symptoms. A characteristically abnormal tracing confirms the presence of cardiac pain, but views on the meaning of a normal tracing need affirming. Whether a strictly normal electrocardiogram can exclude cardiac infarction as the cause of chest pain is a question that calls for an answer, and this paper is meant to contribute to a solution of the problem.

The bipolar CR chest leads have been used deliberately instead of V leads because of their superiority in the investigation of the lesser injuries in the heart following coronary arterial disease; especially does this apply to CR7 where the T wave in health is upright whereas in V7 it is low, sometimes flat, and occasionally inverted.

Scope of the Inquiry

During a period when 1000 consecutive patients with cardiac infarction were examined, 322 other patients were found with like chest pain, but in whom the electrocardiogram seemed normal and where a non-cardiac, and usually dyspeptic, cause was operating. To this latter group 48 such cases have been added since the series with cardiac infarction was completed so that the number with normal tracings is now 370. The progress of all 1370 patients has been watched at intervals of less than two years in order to observe their longevity and the changes that might take place in successive electrocardiograms. In the first group of 1000 patients tracings that might have returned to normal were sought, while in the second group of 370 patients, each with an apparently physiological electrocardiogram, a watch was kept for abnormal changes that might appear subsequently and so would give proof that coronary arterial disease had caused the pain from the start. The period of observation has extended over two years in 829 patients, over four years in 482, over six years in 238, over eight years in 65, and over ten years in 47 patients.

The Electrocardiogram customarily found in Cardiac Pain

The design of the electrocardiogram in the series of patients with cardiac infarction conformed to one of six types, and although these corresponded roughly with the location of the infarct in the isolated examples examined at necropsy, it is known that the electrocardiographic pattern cannot define precisely the exact limits of the infarct. There are a number of reasons for this. First, there is often a natural variation in the course of the affected artery. Secondly, the efficiency of the collateral circulation adjoining the infarcted area is inconstant. Thirdly, the position of the heart varies from its orthodox lie in the chest in different patients. Again, the site of the infarct and its nearness to the base or the apex of the heart affects the electrocardiogram, while the presence of healthy myocardium between the infarct and the electrode will alter its pattern. Lastly, the presence of more than a single area of infarction, old or new, disturbs the tracing. A number of investigators (Parkinson and Bedford, 1928; Whitten, 1930; Barnes and Hall, 1932; Barnes, 1934; Barnes, 1935; Lyon, 1938; Martensen, 1942; Myers *et al.*, 1948*a*, 1948*b*, 1949*a*, 1949*b*, and 1949*c*) have found a measure of agreement when correlating the electrocardiographic with the pathological findings, but since a strict correlation is often wanting, and since the location of most infarcts has no direct influence on prognosis, it is seldom wise to dogmatize on the place of the infarct. Instead, a better custom is to adopt the electrocardiographic classification, and the characteristic patterns for salient infarction are shown in Fig. 1 and for restricted infarction in Fig. 2; the incidence of each kind is shown in Tables I and II.

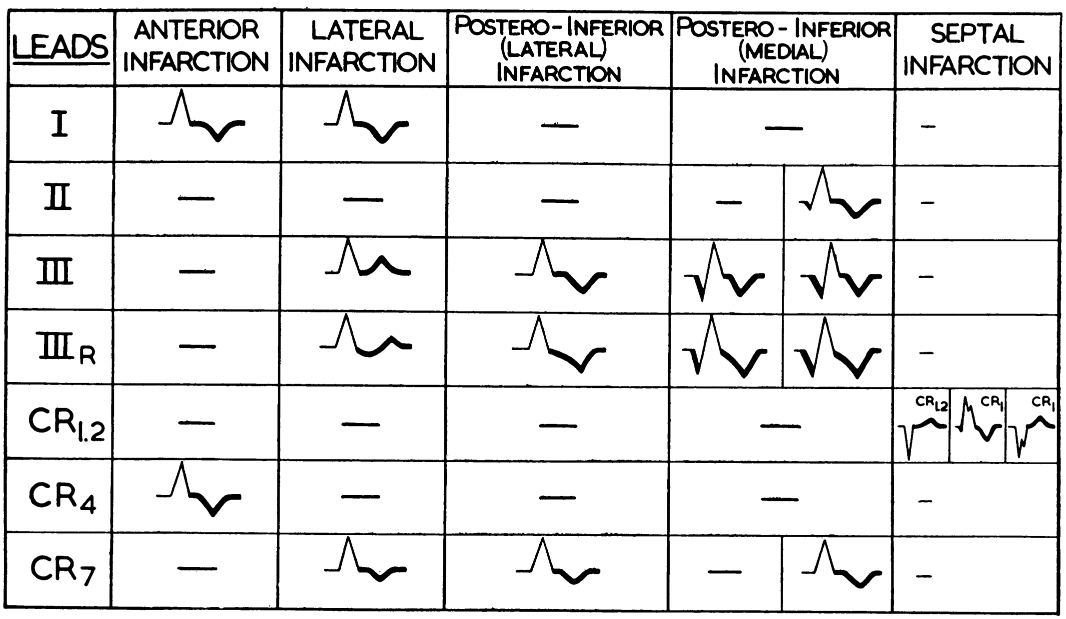

Fig. 1.—Diagrammatic representation of the electrocardiogram in *salient* cardiac infarction according to its situation. Q waves may be added in the anterior and lateral varieties.

The Normal Electrocardiogram in relation to Cardiac Pain

There were 24 instances where the electrocardiogram had been regarded as normal one time and was abnormal another time conforming to the kind found in cardiac infarction. When, however, the tracings in these cases were re-examined in the light of more recent experience of the lesser electrocardiographic changes in cardiac pain (Evans and McRae, 1952) the first opinion on their normality was rescinded in15. The changes that came to light in these cases following this more careful scrutiny included S–T depression, especially in IIIR, in six, low T wave in lead I

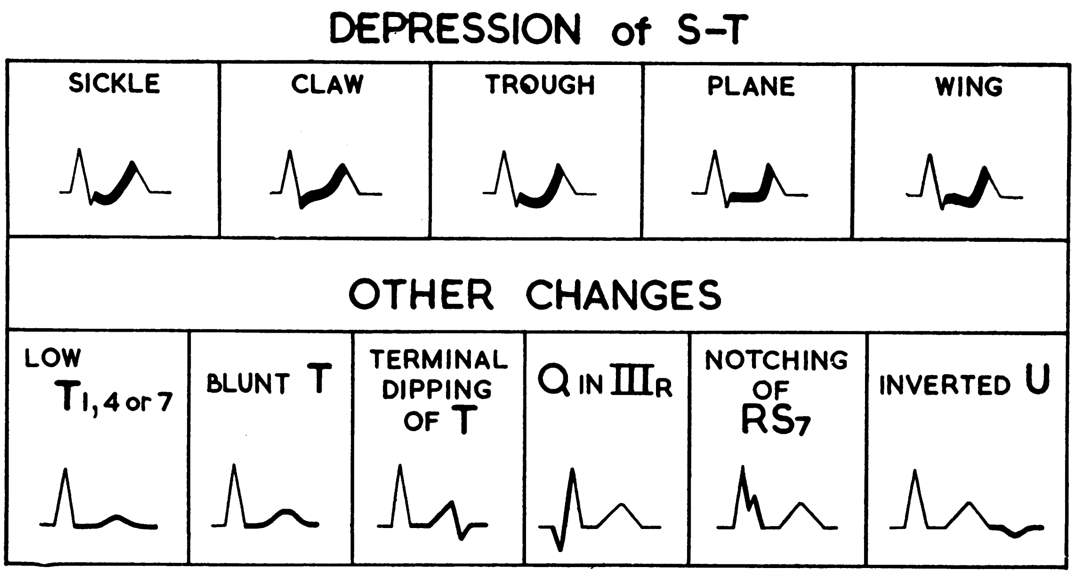

FIG. 2.—Diagrammatic representation of the lesser electrocardiographic changes in *restricted* cardiac infarction.

TABLE I

DISTRIBUTION OF THE INFARCT AND THE MORTALITY RATE AMONG 1000 CONSECUTIVE PATIENTS WITH CARDIAC INFARCTION

Distribution of the infarct		No. of patients	Deaths	
			Number	Mortality rate
Salient	Anterior	234	39	17
	Lateral	126	28	22
	Postero-inferior (lateral)	130	27	20
	Postero-inferior (medial)	132	24	18
	Septal	50	7	14
Restricted		328	60	19
Total		1000	185	19

and in CR4 or CR7 in three, the appearance of a significant Q wave in IIIR in three, a blunt T wave in CR4 in one, splintering of the RS in CR7 in one, and an inverted U wave in one.

There were left, therefore, nine electrocardiograms in patients with cardiac pain which one time showed no departure from the design at present accepted as belonging to a healthy subject, but another time showed the characteristic changes found in cardiac infarction (Table III). The number is very small (9 out of 1004) and this fact alone emphasizes the hesitancy with which a diagnosis of cardiac pain should be applied to a patient where the electrocardiogram fails to support it. Small as the number is, however, it calls for a close search for any special clinical or cardiographic features that might help in the readier recognition of patients in this group.

Eight of the patients were men and there was one woman. Their ages varied from 44 to 67 years and the average age was 52. The site and distribution of the pain was in no way distinctive, so that it was retrosternal in all, spreading into the left arm in four and into both arms in three.

TABLE II

THE INCIDENCE OF THE LESSER ELECTROCARDIOGRAPHIC CHANGES AND THE MORTALITY RATE IN 328 PATIENTS WITH RESTRICTED CARDIAC INFARCTION

Cardiographic sign	No. of patients	Death as sequel to cardiac infarction
Depression of S–T segment	206	35
Low T wave	86	23
Blunt T wave	8	2
Terminal dipping of T wave	5	0
Q developing in IIIR	14	0
Notching of RS segment in CR7	4	0
Inversion of U wave	5	0

TABLE III

THE INCIDENCE OF A NORMAL ELECTROCARDIOGRAM AMONG PATIENTS WITH CHEST PAIN CLINICALLY CHARACTERISTIC OF CARDIAC PAIN

Patients judged to have cardiac infarction (1000)		Patients with cardiac-like pain (370)	
Cardiogram remaining abnormal	Cardiogram returning to normal	Cardiogram remaining normal	Cardiogram initially normal, becoming abnormal
995	5	366	4 *

* These four cases were regarded at first as having cardiac-like pain on the grounds that their electrocardiograms were strictly normal.

Similarly, the character of the pain did not vary from that met with in patients with cardiac infarction, save that the pain seemed more liable to recur at night when it was often intense, lasting an hour or so, but without inducing a state of severe shock. On occasion also the relief of pain that followed exertion was not so instantaneous on resting as is usual in patients with pain from obstructive coronary arterial disease. In none of the nine patients was there hypertension, triple heart rhythm, cardiac enlargement, or pulmonary congestion; indeed, they showed no abnormal signs on clinical and radiological examination of the heart.

In four of the nine cases the normal cardiogram was the initial tracing and in five it was the subsequent one. When the patients were watched after they had shown the abnormal tracing it recovered completely in eight while in one the R wave remained absent in CR1 although the T wave had recovered in all leads.

Among the 1000 patients with cardiac infarction there were 234 who showed a depression or inversion of the T wave in lead I and inversion of the T in CR4, and these changes were accepted as meaning that the myocardial injury was situated for the most part in the anterior wall of the left ventricle (Table I). When the CR1 cardiogram was examined in these patients they were separated into two main groups (Table IV).

In the first group there were 140 patients in whom CR1 was a normal lead. During the time when the progress of these patients was watched there were 28 deaths. In no single instance did the electrocardiogram resume a strictly normal pattern.

In the second group of 94 patients some abnormality was present in CR1 and according to the kind of deformity, these patients were divided into four classes.

In 44 a significant Q wave was the only abnormal finding in CR1 and in none of these did the cardiogram recover; during the period of observation there were seven deaths.

Similarly, among the 26 patients where the CR1 lead showed inversion of the T wave as well

TABLE IV

THE PROGRESS OF ELECTROCARDIOGRAPHIC CHANGES IN 234 PATIENTS WITH DEPRESSION OR INVERSION OF THE T WAVE IN LEAD I AND INVERSION OF THE T IN CR4 IN RELATION TO FINDINGS IN THE RIGHT PECTORAL CARDIOGRAM (CR1)

State of the CR1 cardiogram		No. of patients	Patients showing complete recovery of the whole cardiogram	Deaths
Normal (140)		140	0	28
Abnormal (94)	Deep Q wave	44	0	7
	Q wave present and T wave inverted	26	0	2
	T wave inversion associated with changes in the general electrocardiogram	14	0	1
	Inversion of T wave only	10	8	0

as a Q wave, in none did the electrocardiogram recover completely. In one patient, however, a previous cardiogram had proved to be a normal tracing at a time when chest pain on effort was a symptom, but following the appearance of a Q wave in CR1 and CR2 in addition to inversion of the T wave in these leads and in I and CR4, a significant Q wave remained in CR1 and CR2 although the T waves had recovered (Fig. 3).

In a third group there were 14 patients where CR1 showed inversion of the T wave together with slight changes in the general cardiogram. These changes consisted of depression of the S–T segment in nine, a significant Q wave in leads I, CR4, or CR7 in three, a low T wave in leads I and CR7 in one, and an inverted U wave in one; such blemishes indicated injury to the lateral and/or postero-inferior portions of the left ventricle in addition to the anterior wall. In none of the 14 patients did the electrocardiogram recover completely during the period of observation when one died.

The fourth group held ten patients and in these, inversion of the T wave was the only deformity in the CR1 cardiogram. The electrocardiogram recovered in its entirety in eight out of the ten patients (Fig. 4 to 9). Even in the remaining two cases it had recovered at a subsequent examination except for a Q wave in CR1 in one (Fig. 10) and notching of the RS stem in lead CR7 in the other (Fig. 11). Thus, when inversion of the T wave in CR1 and CR4 was accompanied by significant Q waves or other lesser changes elsewhere in the tracing the cardiogram did not recover completely. Further, when T wave inversion in CR4 was not accompanied by T inversion in CR1 the cardiogram also failed to recover even in the absence of significant Q waves or depression of the S–T segment. Such results show that here is a distinctive electrocardiogram which possesses a remarkable facility to right itself even though at one time its deformity is so patent as to suggest an extensive myocardial injury. It is here proposed that this distinctive tracing in a patient subject to cardiac pain should be named the *mutable electrocardiogram.*

THE DESIGN OF THE MUTABLE ELECTROCARDIOGRAM

All ten patients whose electrocardiograms with two exceptions recovered wholly showed a common pattern. Thus, the T wave was inverted in chest leads CR1 to CR4 and was sometimes flat or inverted in the left ventricular leads as far as CR7; the T was inverted in lead I and occasionally in lead II as well, and it often lost height in IIIR. An abnormal Q wave was always absent in every lead, nor were there any changes customarily found in salient or restricted infarction graphically represented in Fig. 1 and 2. This distinctive electrocardiogram bears no resemblance to the one described for coronary insufficiency by Buchner *et al.* (1935) and by Master *et al.* (1941), in that depression of the S–T segment, regarded here as meaning restricted cardiac infarction, was not a feature of any of the cases.

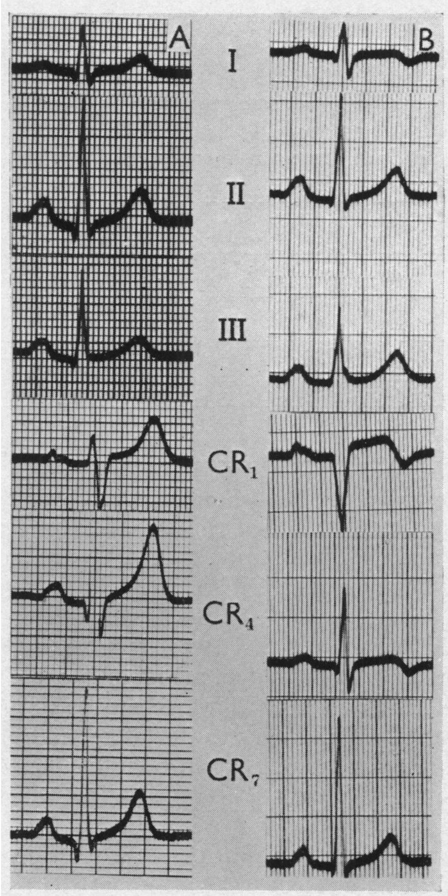

FIG. 3.—One-time normal electrocardiogram in cardiac pain. The tracing (A), recorded in a patient liable to chest pain on effort during the two previous weeks, is normal. (B) was taken a fortnight later and shows inversion of T wave in leads I, CR1, and CR4 as well as a deep Q wave in CR1. Subsequently the T waves recovered, but the significant Q wave remained in leads CR1 and CR2.

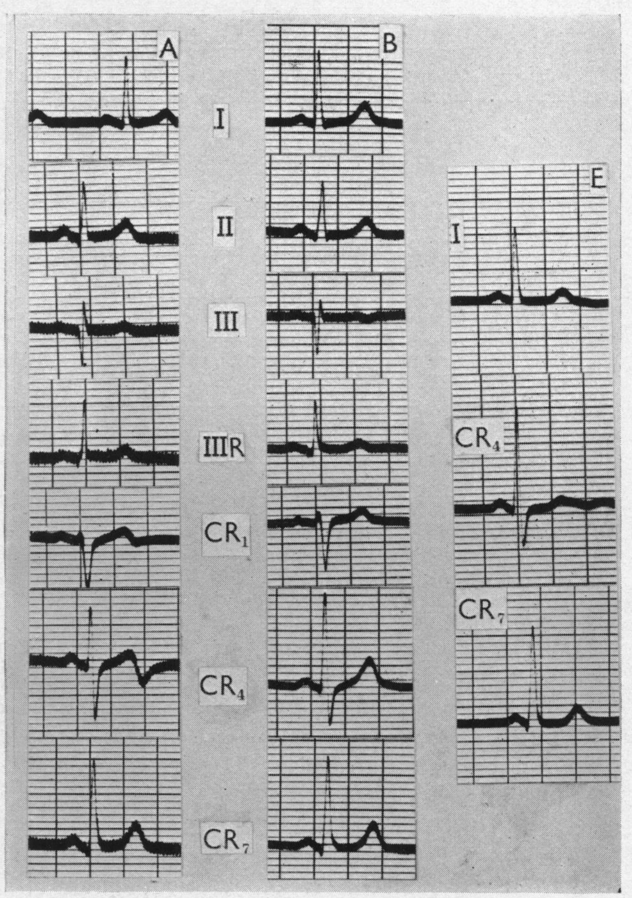

FIG. 4.—*Coronary arteriospasm.* In (A) the T wave is low in I, inverted in CR4, and shows terminal dipping in CR1. (B) recorded four months later is a strictly normal tracing; the exercise electrocardiogram (E) is abnormal.

The importance of *terminal dipping of the T wave* has already received emphasis (Evans and McRae, 1952) for it proved as useful as frank inversion of the T wave in the recognition of a myocardial injury from coronary arterial disease. This change in CR1 was tested to see whether its presence might indicate a special susceptibility to recover. This terminal dipping of the T in CR1 appeared in 5 of the 14 cases where lesser changes in the general tracing occurred in company with the features which characterize the mutable cardiogram; none of these tracings recovered completely. Again, the dipping T wave was present in 3 of the 9 cases where the cardiogram was onetime strictly normal, so that the sign denotes the early age of the myocardial injury, and its presence or its absence does not predict the reversibility or permanence of any abnormal tracing so that in this respect it appears to have the same significance as frank inversion of the T wave.

The effect of *exercise* on the mutable electrocardiogram was sought in the nine patients that exhibited it; the strictly normal tracing became temporarily deformed following strenuous exercise

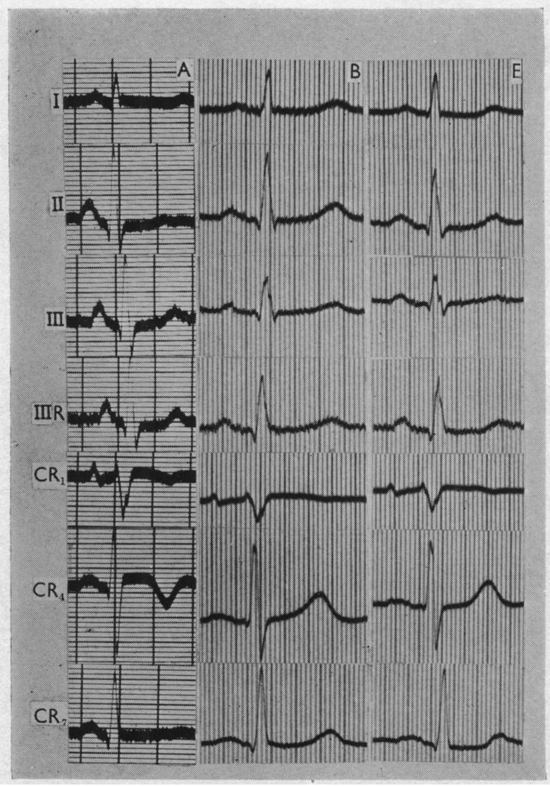

FIG. 5.—*Coronary arteriospasm.* In (A) the T wave is flat in leads I and CR7, low in lead II, and inverted in leads CR1 and CR4. (B) Recorded four years later is a strictly normal tracing; the exercise cardiogram (E) is abnormal.

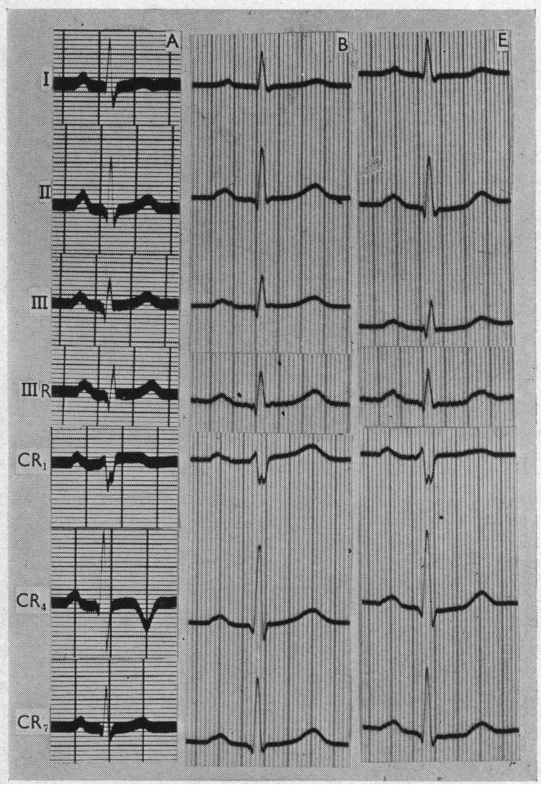

FIG. 6.—*Coronary arteriospasm.* In (A) the T wave shows terminal dipping in CR1, inversion in leads I and CR4, and is low in CR7. (B) Recorded three years later is a strictly normal tracing; the exercise cardiogram (E) is slightly abnormal.

in eight of the nine cases and regained its normal form within ten minutes of discontinuing the exercise. In most of the cases some months had elapsed since a severe paroxysm of pain had overtaken them, although most of them were experiencing periodic episodes of chest discomfort on exertion. In the remaining patient although an exercise electrocardiogram had once showed changes when the resting tracing was normal, a second test carried out when severe chest pain had been absent for over a year failed to produce any blemish in the tracing. It appears, therefore, that the fully recovered mutable cardiogram yields a positive test if this is carried out within six months, but if a longer period has elapsed, provided no strong attacks of pain have visited the patient, strenuous physical exercise may fail to induce a pathological change in the cardiogram even though chest discomfort on walking quickly has continued as a symptom.

THE SIGNIFICANCE OF THE MUTABLE ELECTROCARDIOGRAM

A watch over the progress of patients showing distinctive changes identified with this labile electrocardiogram has established the benignity of the lesion. Over many years in spite of the patients' liability to recurrent attacks of cardiac pain either following effort or while at rest, none died, and with two exceptions where the residual changes were slight, the electrocardiogram recovered completely within a few months of experiencing a severe episode of pain and showed no permanent changes due to frank cardiac infarction.

The characteristic deformity in the tracing indicates a diffuse and intense cardiac ischæmia from interruption of the blood flow in the left coronary artery. The wide distribution of the T wave

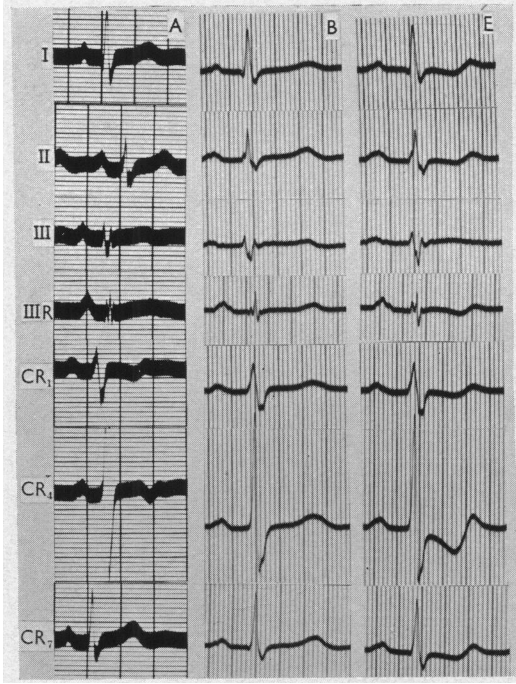

FIG. 7.—*Coronary arteriospasm.* In (A) the T wave is inverted in CR1 and CR4. The tracing (B) recorded a year later is within normal limits; the exercise cardiogram (E) is abnormal.

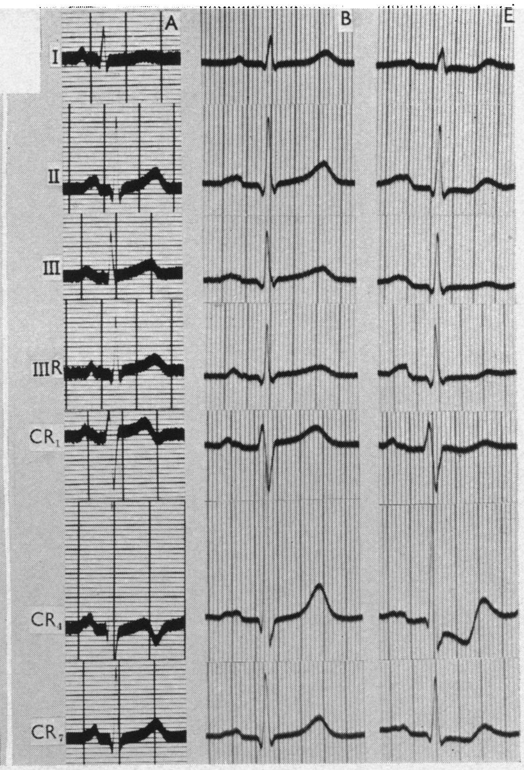

FIG. 8.—*Coronary arteriospasm.* In (A) the T wave is low in lead I, inverted in CR4, and shows terminal dipping in CR1; (B) recorded three years later is a strictly normal tracing; the exercise cardiogram (E) is abnormal.

inversion reaching station CR1 on the right and often as far as CR7 on the left suggests that the artery is involved near its source. Such interruption is short-lived and it does not last long enough to cause death of the patient nor any part of the heart muscle. In time, usually within three to six months and sometimes much sooner, the bruised muscle regains its normal state even if lesser episodes of pain recur in the meantime. This temporary interruption of the coronary blood stream appears to be explained by a spasm of an artery unaffected by material atherosclerosis. Other examples within one's experience appear to exhibit the same mechanism in the presence of coronary arterial disease, when the electrocardiogram has recovered to a remarkable degree, although not wholly (Fig. 12), and in such instances the myocardial injury, restricted to a small area, persists; naturally such a condition cannot claim the good prognosis which characterizes the special group where the recovered cardiogram shows no trace of blemish.

The effect of ingestion of *trinitrin* was not tried in the earlier cases when the electrocardiogram exhibited conspicuous inversion of the T wave because previous experience (Evans and Hoyle, 1933) had failed to give information of practical value from this test. When trinitrin, however, was prescribed while the electrocardiogram was showing a return to a normal pattern, the depressed T wave gained significantly in height (Fig. 13). This reaction also suggests that the T wave deformity in the mutable cardiogram is the outcome of cardiac ischæmia following temporary coronary arteriospasm rather than cardiac infarction from permanent coronary arterial occlusion.

Having regard to the mechanism inducing the cardiac ischæmia and the selective area of its distribution, the actual site of the spasm is in the left coronary artery distal to its circumflex branch

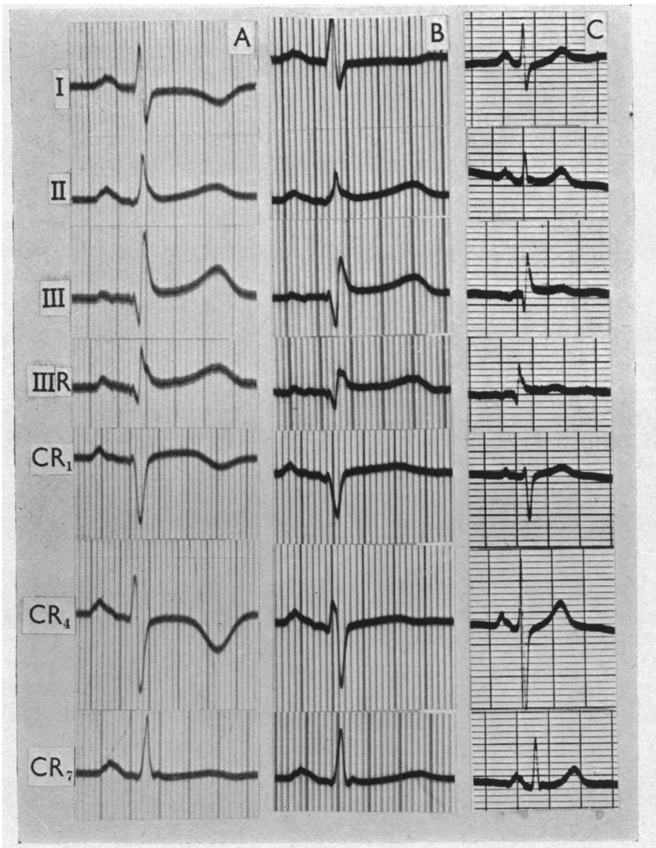

FIG. 9.—*Coronary arteriospasm.* In (A) the T wave is inverted in leads I, CR1, and CR4, and is low in CR7. The changes are slight in (B) recorded four months later, and are absent in (C) recorded two months later.

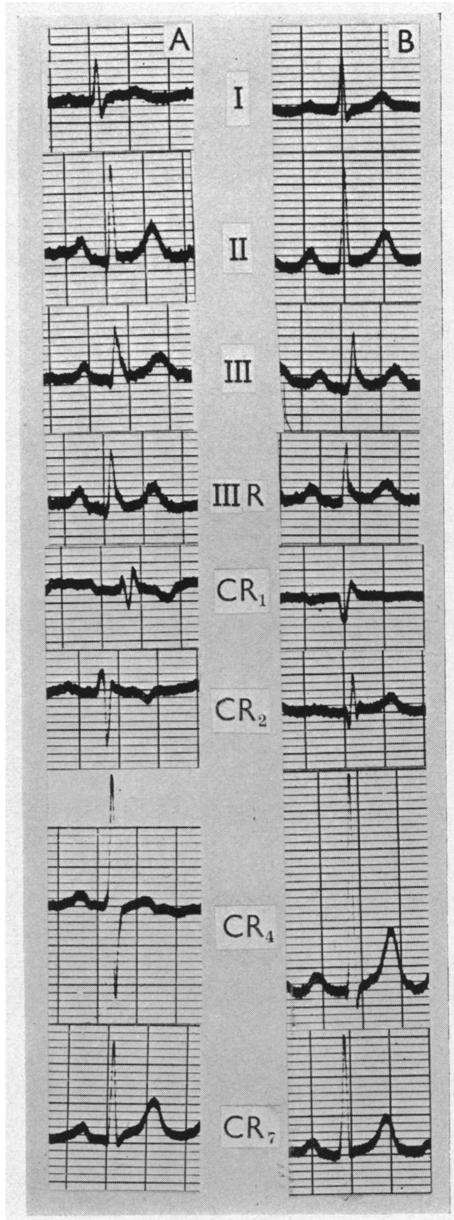

FIG. 10.—*Coronary arteriospasm.* In (A) the T wave is low in lead I and inverted in leads CR1, CR2, and CR4. The tracing (B) recorded two years later remains normal except for the presence of a Q wave in leads CR1 and CR2.

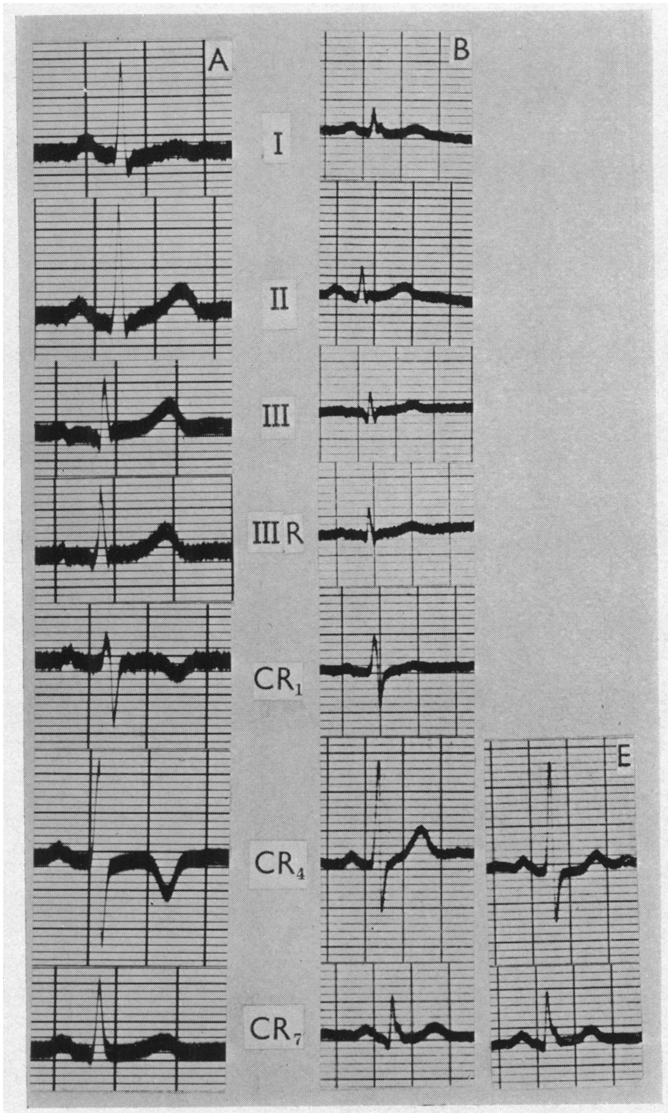

FIG. 11.—*Coronary arteriospasm.* In (A) the T wave is low in leads I and CR7 and inverted in CR1 and CR4. (B) recorded five months later is normal except for notching of the RS segment in CR7; the exercise cardiogram (E) shows no added abnormality.

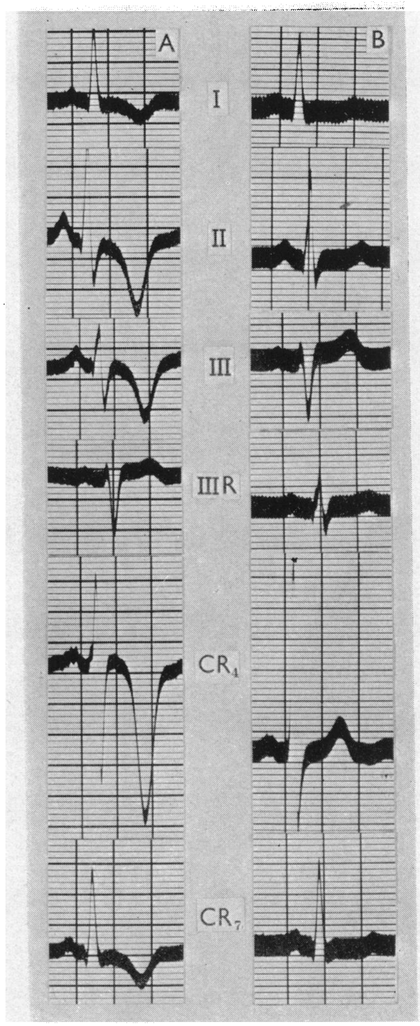

FIG. 12.—*Cardiac infarction.* In (A) the T wave is deeply inverted in leads I, II, III, CR4, and CR7. (B) recorded a year later shows remarkable recovery of the T waves which are low only in leads I, II, and CR7

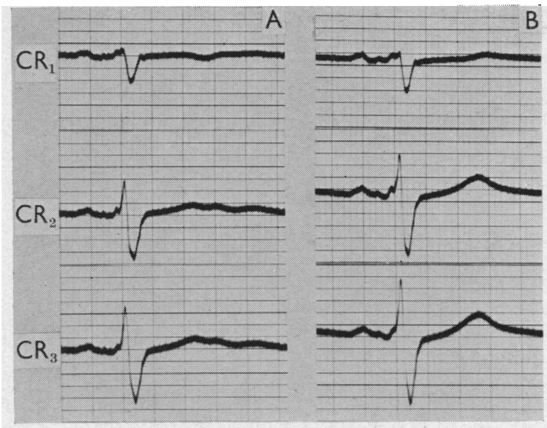

FIG. 13.—*Coronary arteriospasm.* An inverted T wave in CR1 and a low T in CR2 and CR3 in (A) from a patient showing the mutable electrocardiogram are upright in (B) recorded four minutes after chewing 1/25 gr. (2·6 mg.) of glyceryl trinitrate.

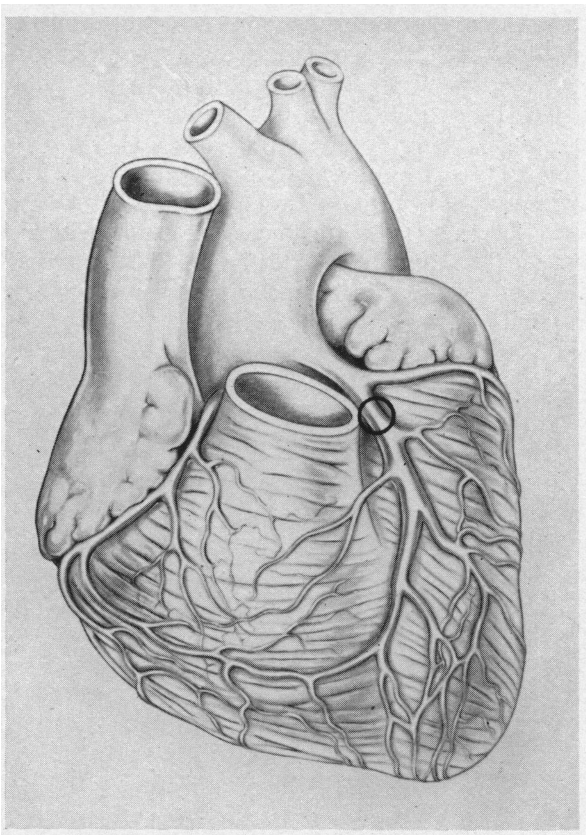

FIG. 14.—*Distribution of the left coronary artery.* The site of the arteriospasm which gives rise to the mutable cardiogram is indicated by a circle and is about 1 cm. proximal to the seat of election for atheromatous obstruction and thrombosis, and lies between the circumflex and the left marginal branches of the left coronary artery.

and proximal to the left marginal branch unless this takes origin from the left circumflex (Fig. 14). No other part of the coronary circulation has been found to be so susceptible to such abrupt and temporary closure for no example of complete recovery of an electrocardiogram has been met with among patients in whom the tracing allocated the injury to either the lateral or postero-inferior portions of the left ventricle. This distinctive segment of the coronary circulation possessing a special aptitude to spasm might be named the *coronary floodgate* for it can suddenly close and interrupt the blood supply to a large area of the heart and as readily it can open and re-establish the circulation.

It was late in the investigation that special attention was paid to the possible existence in the same patient of a like-spasm in situations other than the coronary circulation. Two possible examples of this were met with, one involving the retinal artery and the other the transverse colon, but as such episodes might have been fortuitous, they do not in the meantime allow one to draw firm conclusions from this association.

East and Oram (1948) published a valuable paper in which they recorded 34 cases of cardiac pain where a remarkable degree of recovery of the deformed T wave had taken place. They described 10 of these in detail and showed the electrocardiograms in nine. A right pectoral lead, however, was only available in one patient (their Case 3) and the tracing in this instance recovered completely except that the T wave in V1 remained slightly inverted; this demonstrates the inferiority of lead V1 with its tendency to show an inverted T in health, compared with CR1 in the investigation of these cases. In the bipolar lead, should deformity of the T wave remain, it would suggest that complete recovery of the ischæmic electrocardiogram had not taken place, although in the patients described here the deformity in the CR1 tracing usually disappeared earlier than the abnormality in CR4. Complete recovery of the electrocardiogram also took place in two other patients in East and Oram's series (their Cases 2 and 7), but in neither was the right pectoral lead recorded. In their remaining six cases recovery of the tracing was only partial and signs remained that indicated a residual myocardial injury. They, too, considered that spasm of the coronary artery had caused the temporary electrocardiographic changes. Thompson (1952) published three cases of cardiac pain to illustrate favourable results from treatment with anticoagulants. One of his patients (Case 2) showed a tracing identical in pattern with the mutable electrocardiogram described here; the tracing returned to normal and remained so during a period of three years when the patient had also kept free from symptoms.

Conclusion

Doubt has remained on the true significance of a strictly normal electrocardiogram in a patient in whom the diagnosis of cardiac pain is suspected. The time is not long past when a normal tracing in a patient whose pain apparently was caused by cardiac infarction, occasioned no surprise, but since the lesser electrocardiographic changes identified with coronary arterial disease have gained more general recognition the incidence of a normal cardiogram in such patients has decreased.

The question whether the electrocardiogram of cardiac infarction is ever normal still needs to be answered. The present investigation supports the view that it is never strictly normal provided the tracing has been a competent one, and provided the mutable electrocardiogram described here and signifying coronary angiospasm is recognized. When cardiac pain arises from such spasm the tracing becomes grossly abnormal from the ensuing cardiac ischæmia, always preserving a consistent pattern, and thereafter gradually returning to assume its previous normal design even though light attacks of cardiac pain continue to recur.

The mutable electrocardiogram is characterized by inversion of the T wave in chest leads CR1 to CR4 and often beyond this as far as station CR7; early on, the deformity may be confined to the terminal portion of the T wave which may dip sharply below the iso-electric level. The T wave is usually inverted in lead I as well Signs of a permanent myocardial injury from coronary arterial disease like a significant Q wave or depression of the S–T segment are absent from this distinctive cardiogram. When the tracing has recovered, strenuous physical exercise will re-introduce the deformity as a rule unless the period of immunity from pain has been a long one. Only 10 such tracings were found among the 1370 patients whose symptoms suggested cardiac pain, and it is significant that the eight cardiograms which recovered completely were from this number, while only a small blemish remained in the other two tracings.

Because of the favourable prognosis associated with the mutable electrocardiogram, denoting as it does coronary arteriospasm with temporary cardiac ischæmia rather than lasting coronary occlusion with cardiac infarction, its recognition is a matter of considerable moment to the occasional patient who may exhibit it.

Sir John Parkinson made valuable suggestions on the preparation of this paper.

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